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FULL RESEARCH PAPER

Development of ascochyta blight (*Ascochyta rabiei*) in chickpea as affected by host resistance and plant age

A. K. Basandrai · D. Basandrai · S. Pande · M. Sharma · Sanjay K. Thakur ·
H. L. Thakur

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Abstract Ascochyta blight caused by *Ascochyta rabiei*, is the most destructive disease in many chickpea growing countries. Disease development varies with the growth stage and host resistance. Hence, disease development was studied in cvs ICCX 810800 (resistant), ICCV 90201 (moderately resistant), C 235 (moderately susceptible), ICCV 96029 and Pb 7 (susceptible) under controlled environment (ICRISAT, Patancheru) and field conditions (Dhaulakuan, Himachal Pradesh) at seedling, post-seedling, vegetative, flowering and podding stages. Under controlled environment, the incubation period and terminal disease reaction (TDR) did not vary significantly at different growth stages against

virulent isolate AB 4. Cultivars ICCX 810800, ICCV 90201 and C 235 showed a significantly longer incubation period than the susceptible cv. Pb 7. Cultivar ICCX 810800 showed slow disease progress and the least TDR. Field experiments were conducted during the 2003–2004 and 2004–2005 growing seasons. During 2003–2004, TDR was higher in plants inoculated at podding and the flowering stage and the lowest disease reaction was recorded in ICCX 810800. A severe epidemic during 2004–2005 was attributed to the favourable temperature, humidity and well distributed high rainfall. TDR did not differ significantly at any of the growth stages in susceptible cvs ICCV 96029 and Pb 7. With respect to seeding date and cultivar, the highest yield was recorded in the early-sown crop (1,276.7 kg ha⁻¹) and in ICCV 90201 (1,799.3 kg ha⁻¹), respectively. The yields were greatly reduced in all the cultivars during 2004–2005 and the highest yield was recorded in ICCX 810800 (524.7 kg ha⁻¹). Integrated disease management using resistant cultivars, optimum sowing period and foliar application of fungicides will improve chickpea production. The experiment under controlled environment and field conditions (during the epidemic year) showed a similar disease development.

A. K. Basandrai (✉) · D. Basandrai ·
S. K. Thakur · H. L. Thakur
Hill Agricultural Research and Extension Centre,
Choudhary Saravan Kumar Himachal Pradesh
Agricultural University, Dhaulakuan, Himachal Pradesh
173 001, India
e-mail: bunchy@rediffmail.com

S. Pande · M. Sharma
International Crops Research Institute for the Semi-Arid
Tropics, Patancheru, Andhra Pradesh 502 324, India

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Plant growth stage · Resistance

Introduction

Chickpea is world's third most important grain legume. It is a major source of dietary protein and a significant contributor to agricultural sustainability by fixing atmospheric nitrogen. It diversifies agricultural production systems in rotation with cereals. During the year 2004–2005, the world chickpea production was approximately 8.58 million tonnes from an area of approximately 11.16 million hectares (Ikisan 2000). The seed yield varies from <390 to 3,600 kg ha⁻¹ depending upon environmental conditions and management for biotic and abiotic constraints. Ascochyta blight, caused by *Ascochyta rabiei*, is a major factor in the low productivity of chickpea in various countries of western Asia and north Africa, the northwestern plains in the Indian subcontinent, Australia, North America, Latin America and southern Europe (Gan et al. 2006; Nene and Reddy 1987; Pande et al. 2005). It infects during all growth stages of plants where temperature and rainfall are favourable for pathogen development (Pande et al. 2005, Shtienberg et al. 2000) and may cause yield losses up to 100%. The disease can be managed by the cultivation of resistant cultivars. Plant age had been reported to have no impact on disease resistance in some cultivars (Trapero-Casas and Kaiser 1992) whereas, in others it has been reported to decline with plant maturity (Chongo and Gossen 2001; Gan et al. 2006; Nene and Reddy 1987; Singh and Reddy, 1993). This change from resistance to susceptibility with maturity refutes the importance of resistance as the main strategy for managing this disease. In this context, present studies were undertaken to study development of ascochyta blight as affected by plant age, environmental factors and resistance status of some Indian cultivars.

Materials and methods

Host

Five desi chickpea cultivars; C 235, ICCV 90201, ICCX 810800, ICCV 96029 and Pb 7 were used in the present studies. The pedigree, origin and resistance status of these cultivars is given in Table 1. Cultivar Pb 7, an old cultivar from Punjab (India) and ICCV 96029 were highly susceptible to ascochyta blight. Cultivar ICCV 96029 is a very early maturing and cold tolerant line suitable for contingent crop planning in the northwestern plain and hill zone of India. ICRISAT lines ICCX 810800 and ICCV 90201 have been released in Himachal Pradesh (India) for cultivation as ascochyta blight and Fusarium wilt-resistant cultivars. Cultivar C 235 is an old and widely adapted variety released in many countries.

Pathogen

Single conidial isolates of *A. rabiei*, AB 4 (isolated from infected plants at Hissar, Haryana) and isolate AB 6 (isolated from infected plants at Dhaulakuan, Himachal Pradesh) were used for the controlled environment and field studies, respectively. Isolate AB 04 was highly virulent whereas, isolate AB 06 was moderate in virulence (Basandrai et al. 2005). The isolates were multiplied on chickpea dextrose agar medium for 15 days and used for the studies.

Controlled environment studies

The experiment was conducted in the growth chambers at the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru.

Table 1 Pedigree, origin and resistance status of chickpea cultivars

Cultivar	Pedigree	Origin	Reaction to ascochyta blight
ICCX 810800	GL 769 × ILC 202	ICRISAT	Resistant
ICCV 90201	GL 769 × ICC 1069	ICRISAT	Moderately resistant
C 235	C 1235 × IP 58	PAU, Ludhiana	Moderately susceptible
ICCV 96029	ICCV 2 × ICCV 93929	ICRISAT	Highly susceptible
Pb 7	ICC 4991	A local selection from Punjab	Highly susceptible

Plant growth conditions

Plants of the test cultivars were raised in 25 cm diameter plastic pots filled with a mixture of sterilized sand and vermiculite (10:1), in a greenhouse maintained at $25 \pm 3^\circ\text{C}$ and a 12–13 h photoperiod under natural light. Staggered sowing was done for 8 weeks to produce plants that were 2–9 weeks of age representing five distinct growth stages (Table 2) at the time of inoculation. Five plants were maintained in each pot with three replications.

Inoculation and incubation

The pots with plants of different growth stages were transferred to the growth chamber maintained at $20 \pm 1^\circ\text{C}$ and light intensity of 1,500–1,600 lux using artificial daylight fluorescent tubes. The inoculum was mass-multiplied on Kabuli chickpea seeds. Seeds were soaked overnight in water and about 50 g of these seeds were transferred in 250 ml flasks. These were sterilized by autoclaving at 121°C (15 psi) for 25 min. Highly sporulating inoculum of the isolate AB 4, grown on chickpea dextrose agar, was transferred aseptically onto the seeds in the flask. The inoculated flasks were incubated at $20 \pm 0.5^\circ\text{C}$ with a 12 h alternate light and dark period. The flasks were frequently shaken to avoid clumping of inoculum. Abundant conidial production was obtained after 6–8 days. The conidia were harvested in sterilized water. The plants were inoculated by spraying a suspension of isolate AB 4 (5×10^4 conidia ml^{-1}) in water. The inoculated plants were allowed to dry for 4 h and thereafter incubated at 100% continuous RH for 6–7 days.

Table 2 Growth stage of chickpea cultivars at which inoculations were done

Age in weeks	Growth stage	Growth stage description
2	I	Seedling
3	II	Post-seedling: Branch initiation
4–5	III	Vegetative: Branching continue- Floral bud initiation
6–7	IV	Flowering: Flowering and stem hardening
8–9	V	Podding: Flowering to pod formation

Data recording

The plants were observed daily to determine incubation period i.e. the period (days) from inoculation to appearance of first visible symptoms. Thereafter, the data were recorded for disease reaction on alternate days for each plant in the pot on a 1–9 scale (Nene et al. 1981). These data were used to determine the dynamics of disease progress.

Field studies

Field trials were conducted in the experimental fields at the Choudhary Saravan Kumar Himachal Pradesh Agricultural University, Hill Agricultural Research and Extension Centre, Dhaulakuan, India, a hot spot for ascochyta blight, during 2003–2004 and 2004–2005. The test cultivars were planted in 0.9×3 m plots with row-to-row and plant-to-plant spacing of 30 and 10 cm, respectively in a split-plot design, with date of sowing as the main plot and varieties as sub-plots. Genotype ICCV 96029 was also included in the field studies. The first planting was done on 24 October during both years and subsequently, four more staggered plantings were done fortnightly to produce plants at five different growth stages, viz. seedling (I), post-seedling, branch initiation (II), vegetative (III), flowering (IV) and podding stage (V). The plots were inoculated by frequently spraying conidial inoculum of isolate AB 6 (10^6 conidia ml^{-1}), mass-multiplied on Kabuli chickpea seeds, starting 4–6 weeks after the last seeding when the plants of all growth stages were available. It was repeated at four-day intervals. In all, 4–5 inoculations were carried out. Ascochyta blight-infected debris was also broadcast in each plot along with the first spray to encourage uniform development of the disease and to prevent disease escape. A Perfo-spray system was used to provide humidity on the dry days between 11.00 h and 17.00 h for 20–30 min every 3 h.

Data recording

The data were recorded on 10 randomly selected plants for terminal disease reaction (TDR) on 1–9 scale (Nene et al. 1981) and yield (kg ha^{-1}) during both years. TDR was also assessed at 2, 4, 6, 8, 10

and 12 weeks after inoculation during 2004–2005 and was used to determine the dynamics of disease progress. Analyses of variance were done using the CPCS1 computer programme.

Results

Controlled environment studies

The data recorded for incubation period and TDR under controlled environmental conditions are given in Tables 3 and 4.

Incubation period

The incubation period on the susceptible cv. Pb 7 was the shortest among the cultivars in the trial (3.0 days). Incubation period in cvs ICCX 810800, ICCV 90201 and C 235 was statistically longer compared with the susceptible cv. Pb 7. Cultivar ICCX 810800 showed the longest incubation period (6.4 days), significantly longer than the moderately resistant (ICCV 90201)

and moderately susceptible (C 235) cultivars. The incubation period of the test cultivars did not differ significantly among the different growth stages.

Dynamics of disease development

The disease progress in the test cultivars at different growth stages is presented in Figs. 1–4.

In cv. C 235, the slowest disease progress was recorded in plants inoculated at flowering stage followed by plants inoculated at the post-seedling stage (Fig. 1). In cv. ICCX 810800, the plants at the seedling stage recorded the slowest disease progress (Fig. 2). In cv. ICCV 90201, the slowest disease progress was recorded in plants inoculated at the post-seedling stage followed by plants inoculated at the seedling stage (Fig. 3). The dynamics of disease progress in cv. Pb 7 was similar at all the growth stages (Fig. 4).

Terminal disease reaction

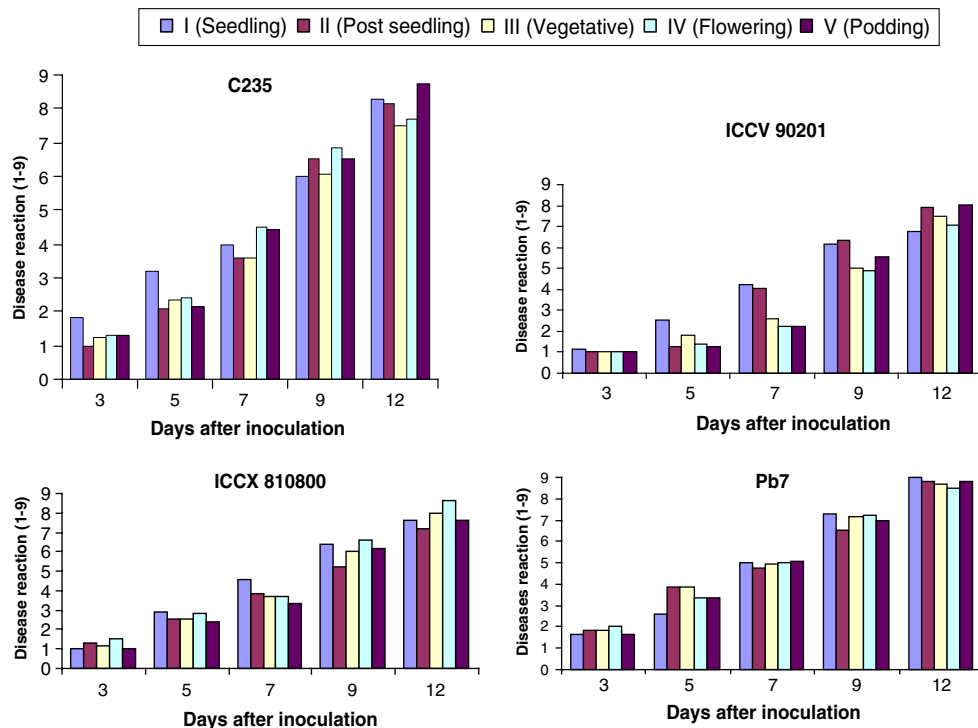
Cultivars ICCX 810800, C 235, ICCV 90201 and Pb 7 developed a TDR of 6.8–8.1, 8.0–8.7, 7.2–8.5 and 8.5–9.0, respectively in plants inoculated at different

Table 3 Effect of growth stage and cultivar on incubation period of ascochyta blight infection under controlled environmental conditions

Cultivar	Incubation period (days) at growth stage					Mean
	I	II	III	IV	V	
C 235	4.00	4.00	4.33	4.33	4.33	4.20
ICCX 810800	6.67	6.67	6.33	6.67	5.67	6.40
Pb 7	3.00	3.00	3.00	3.00	3.00	3.00
ICCV 90201	4.00	4.33	4.00	4.67	5.33	4.47
Mean	4.42	4.50	4.42	4.67	4.58	
CD (5%)	Cultivar = 0.52, Plant age = NS, Cultivars \times Plant age = NS					

Table 4 Terminal disease reaction of chickpea cultivars against *A. rabiei* inoculated at different growth stages under a controlled environment

Cultivar	Disease reaction (1–9) at growth stages					Mean
	I	II	III	IV	V	
C 235	8.7	8.4	8.0	8.3	8.3	8.3
ICCX 810800	6.8	7.3	7.5	7.1	8.1	7.3
Pb 7	9.0	8.8	8.7	8.5	8.8	8.8
ICCV 90201	7.3	7.2	8.3	7.6	8.5	7.8
Mean	8.0	7.9	8.1	7.9	8.4	
CD (5%)	Cultivar = 0.44, Plant age = NS, Cultivars \times Plant age = NS					



Figs. 1–4 Dynamics of disease development against *Ascochyta rabiei* isolate AB 04 in chickpea cvs C 235, ICCV 90201, ICCX 810800 and Pb 7 at different growth stages (I Seedling;

II Post-seedling; III Vegetative; IV Flowering and V Podding stage) under controlled environmental conditions at ICRISAT, Patancheru

growth stages (Table 4). The mean TDR was non-significant among plants inoculated at different growth stages, but it differed significantly among cultivars. Cultivar ICCX 810800 developed the lowest TDR (7.3) followed by ICCV 90201 (7.8). ICCX 810800 showed the lowest (6.8) TDR at the seedling stage. The resistant cv. ICCX 810800 and the moderately resistant cv. ICCV 90201 had a longer incubation period, slower disease development and the least TDR in plants inoculated at the younger stage and thus showed rate-reducing resistance.

Field studies

Blight appeared in epidemic form during 2004–2005 and it was moderate during the 2003–2004 growing season. All of the cultivars developed the lowest TDR in plants inoculated at the seedling to vegetative stage and the TDR increased consistently in plants at later growth stages (Table 5) during the 2003–2004 growing season. With regard to cultivar averaged

over growth stages, the highest and the lowest TDR were recorded in cvs ICCV 96029 (6.1) and ICCX 810800 (2.2), respectively. With regard to growth stage averaged over cultivars, the highest and the lowest TDR values were recorded in the plants inoculated at the podding stage (5.3) and the seedling stage (2.9), respectively. In cv. ICCX 810800, TDR was the highest (4.4) in plants inoculated at the flowering stage and it differed significantly from plants inoculated at other growth stages. During the 2004–2005 growing season, the TDR was not statistically significant with respect to growth stage and the cultivar \times growth stage interaction. However, the TDR differed significantly among cultivars. The highest TDR was recorded in cv. Pb 7 (8.9) followed by ICCV 96029 (8.8) and, averaged over the growth stages, cv. ICCX 810800 showed the lowest TDR (2.9) followed by ICCV 90201 (4.3).

The effect of ascochyta blight on yield of chickpea cultivars in plants inoculated at different growth stages are summarized in Table 6. In general, the

Table 5 Terminal disease reaction (TDR) of *Ascochyta rabiei* on chickpea cultivars inoculated at varying growth stages under field conditions at Dhaulakuan during 2003–2004 and 2004–2005

Cultivar	Terminal disease reaction (1–9) on plants inoculated at growth stages											
	2003–2004						2004–2005					
	I	II	III	IV	V	Mean	I	II	III	IV	V	Mean
C 235	2.6	2.6	3.3	4.4	4.4	3.5	6.5	5.5	5.7	6.3	5.3	5.9
ICCV 90201	2.1	2.6	2.6	3.2	3.7	2.8	4.2	4.6	4.0	4.6	4.3	4.3
ICCV 96029	4.8	4.8	5.7	6.4	8.8	6.1	8.8	9.0	8.1	9.0	8.9	8.8
ICCX 810800	1.2	1.3	2.0	4.4	2.2	2.2	1.8	2.9	3.1	3.2	3.7	2.9
Pb 7	3.9	4.0	3.9	6.8	7.5	5.2	9.0	9.0	8.7	9.0	8.7	8.9
Mean	2.9	3.1	3.5	5.0	5.3		6.1	6.2	5.9	6.4	6.2	
CD (5%)	Cultivar = 0.48, Growth stage = 0.65, Cultivar \times growth stage = 1.1						Cultivar = 0.5, Growth stage = NS, Cultivar \times growth stage = NS					

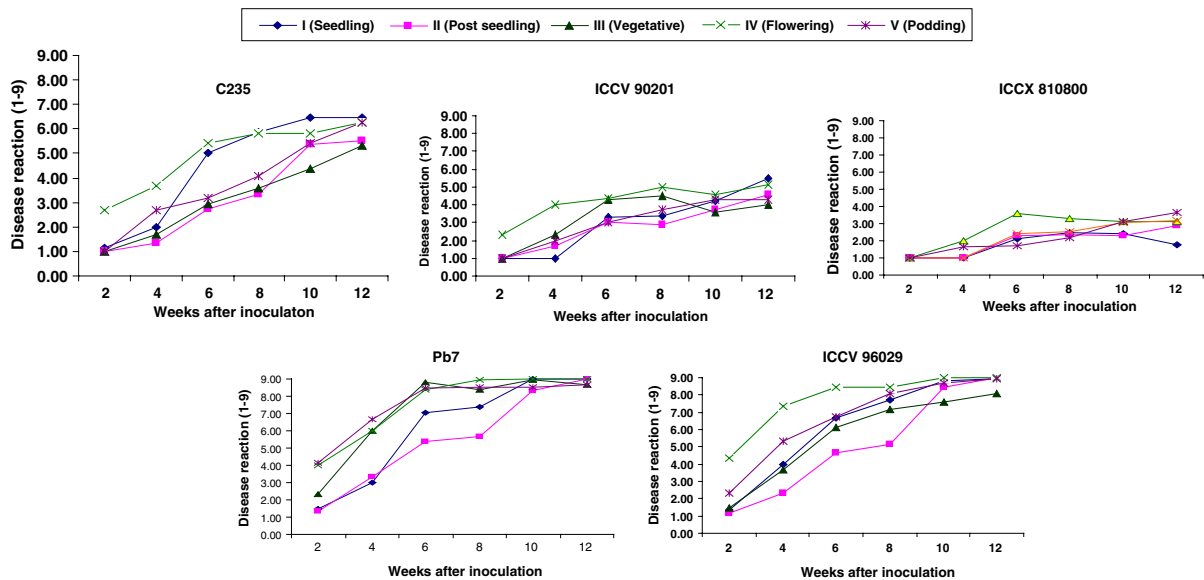
yield was higher during 2003–2004 as compared with the epidemic year 2004–2005. Averaged across the inoculation treatments at various growth stages, the highest yield was recorded in moderately resistant cv. ICCV 90201 (1,799.3 kg ha⁻¹) followed by C 235 (1,259.5 kg ha⁻¹). Averaged across cultivars, the highest yield (1,276.7 kg ha⁻¹) was recorded in the earlier-sown crops (inoculated at the podding stage) and yield decreased consistently with delay in the sowing (Table 6). However, in the very early cv. ICCV 96029, the highest yield (600 kg ha⁻¹) was recorded in late sown crop (inoculated at the post-seedling stage). Yield for this inoculation treatment was similar to that of the crop inoculated at the vegetative stage (563 kg ha⁻¹).

During the 2004–2005 growing season, the highest yield was recorded in cv. ICCX 810800 (524.2 kg ha⁻¹) averaged across sowing dates and in crops sown earlier and inoculated at the podding stage, when averaged across cultivars. In cvs ICCX 810800 and ICCV 90201, the highest seed yield was obtained in the earlier-sown crop (1,204.8 and 307.0 kg ha⁻¹, respectively). Yield decreased drastically in the delayed sowings. Negligible yield was obtained from the susceptible cvs Pb 7 and ICCV 96029.

The dynamics of disease development in cultivars inoculated at different growth stages during 2004–2005 are shown in Figs. 5–9. In cv. C 235, disease appeared earlier and progressed faster in plants

Table 6 Effect of ascochyta blight infection on yield (kg ha⁻¹) of chickpea cultivars sown at different dates at Dhaulakuan during 2003–2004 and 2004–2005

Cultivar	Yield (kg ha ⁻¹) in plants inoculated at growth stage											
	2003–2004						2004–2005					
	I	II	III	IV	V	Mean	I	II	III	IV	V	Mean
C 235	1,251.9	1,084.1	1,353.0	856.7	1,751.9	1,259.5	89.6	20.7	18.9	57.4	254.4	88.2
ICCV 90201	1,107.4	1,203.7	1,024.8	2,723.3	2,937.0	1,799.3	168.1	83.7	174.1	232.6	307.0	193.1
ICCV 96029	444.4	600.0	563.0	113.7	53.7	355.0	7.8	7.8	32.6	71.9	23.3	28.7
ICCX 810800	64.1	555.6	387.8	1,254.4	1,281.5	708.7	130.0	130.0	368.9	787.4	1,204.8	524.2
Pb 7	37.0	403.7	340.7	74.1	359.3	243.0	7.8	7.8	0	7.8	7.4	6.2
Mean	581.1	769.3	733.7	1,004.4	1,276.7		80.7	50	118.9	231.4	359.4	
CD (5%)	Cultivar = 40.0, Growth stage = 37.8, Cultivar \times growth stage = 84.4						Cultivar = 19.7, Growth stage = 10.7, Cultivar \times growth stage = 43.0					



Figs. 5–9 Dynamics of disease development against *Ascochyta rabiei* isolate AB 06 in chickpea cvs C 235, ICCV 90201, ICCX 810800, Pb 7 and ICCV 96029 at different

growth stages (I Seedling; II Post-seedling; III Vegetative; IV Flowering and V Podding stage) under field conditions at Dhaulakuan

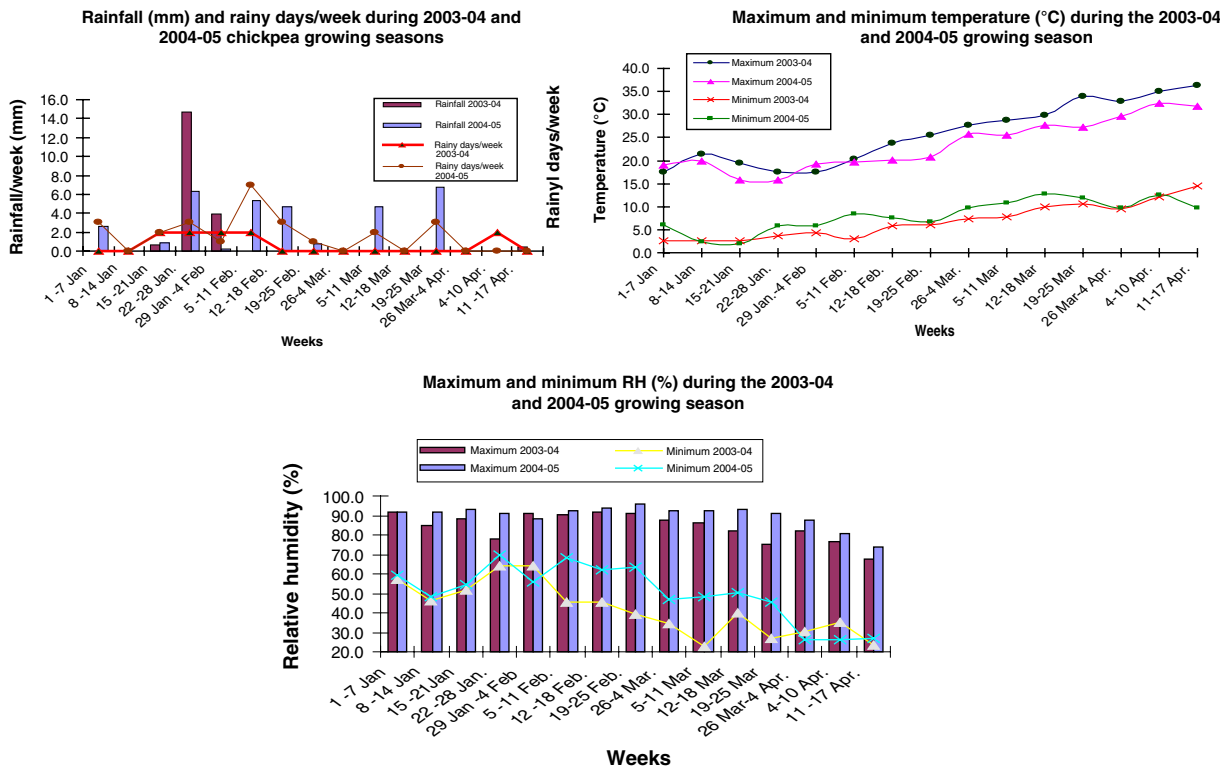
inoculated at flowering, followed by plants inoculated at the seedling stage (Fig. 5). In ICCV 90201, the disease appeared earlier and progressed faster in plants inoculated at the flowering stage, followed by plants inoculated at the podding stage (Fig. 6). In susceptible cultivars, symptoms appeared 2 weeks after inoculation for treatments inoculated at the vegetative stage or later, and 4 weeks after inoculation for plants inoculated at the seedling or post-seedling stages. In contrast, symptoms in cv. ICCX 810800 appeared 4 weeks following inoculation of plants at the vegetative and podding stages and at 6 weeks following inoculation of plants at the seedling, post-seedling and flowering stages (Fig. 7). The disease progressed at a faster rate in plants inoculated at the podding and vegetative stages and progressed at the slowest rate in plants inoculated at the seedling stage.

In susceptible cvs ICCV 96029 and Pb 7, the disease appeared earlier and progressed more quickly at all growth stages, with a TDR of 8.1–9.0 (Fig. 8 and 9).

Discussion

The effect of growth stages on development of ascochyta blight was studied in cultivars with varying

levels of resistance under controlled environment and field conditions. Under the controlled environment conditions, symptoms developed earlier in susceptible cv. Pb 7 with an incubation period of 3.0 days. The incubation period was statistically longer in resistant (ICCX 810800), moderately resistant (ICCV 90201) and moderately susceptible (C 235) cultivars. It was the least at podding stage in cv. ICCX 810800. The incubation period in moderately resistant cv. ICCV 90201 and moderately susceptible cv. C 235 also differed significantly compared with the susceptible cv. Pb 7. Similarly, TDR was also statistically the lowest in cv. ICCX 810800 and it was numerically lower at the seedling stage. This may be because in resistant cultivars, old tissues become more vulnerable to infection than new growth (Chongo and Gossen 2001). Cultivar ICCX 810800 showed a high level of resistance at the seedling to vegetative stage which declined at the flowering to podding stage under controlled environment and field conditions during the epidemic year. These results support earlier studies (Chongo and Gossan 2001; Nene and Reddy 1987; Singh and Reddy 1993) that showed increased ascochyta blight susceptibility as the plant matured. The increased susceptibility in older plants of resistant cv. ICCX 810800 may be due to developmental gene expression, as resistance genes may be highly



Figs. 10–12 Maximum and minimum temperature (°C); rainfall intensity (mm) and distribution (rainy days/standard week) and mean maximum and minimum RH (%) during the cropping season 2003–2004 and 2004–2005

expressed during the seedling to vegetative stage rather than at maturity. This differential response of resistance at different growth stages may be due to the increased secretion of maleic acid (Singh and Sharma 1998), activity of enzymes namely chitinase and exochitinase (Nehra et al. 1997), phytoalexins, namely medicarpin and maackianin and their biosynthetic bio-enzymes, lytic protein enzymes and other PR proteins (Hanselle and Barz 2001).

Plant growth stage had no effect on disease progress and TDR in highly susceptible cvs Pb 7 and ICCV 96029, and these were severely blighted at all growth stages under controlled environment and field conditions during epidemic year 2004–2005. These results were supported by earlier studies (Chongo and Gossen 2001; Trapero-Casas and Kaiser 1992) that showed that growth stage had no effect on disease development in susceptible cultivars.

In the field experiments, substantial differences were observed in TDR among the test cultivars. Characteristic symptom expression, pycnidial fruiting bodies in concentric rings, was more pronounced in

adult plants (8–9 weeks-old) in the field whereas in the growth chamber and in plants at an earlier stage the disease appeared as water-soaked lesions.

During the year 2003–2004, the moderately resistant cv. ICCV 90201 gave the highest yields in the earlier-sown crop and declined with the delay in sowing. This supported earlier studies that showed early-sown moderately resistant cultivars produced a 15–300% higher yield than those sown late (Gan et al. 2002; Siddique and Sedgley 1986). This may be because sowing at the optimum time resulted in the maximum use of available resources and the plants were subjected to fewer stresses (Gan et al. 2002; Siddique and Bultynck 2004). Regardless of blight infection, delayed sowing resulted in lower grain yields as delayed sowing may not have allowed adequate grain filling prior to crop maturity (Gan et al. 2006). In contrast, yield of cv. ICCV 96029 increased with the delay in sowing and the highest yield was obtained when the crop was sown in mid-December. ICCV 96029 is a super early cultivar which flowered in 50–52 days. The earlier-sown crop

(sown 24 October 2003) flowered by mid-December, when the minimum temperature was $<5^{\circ}\text{C}$, which resulted in lower pollen viability and embryo abortion, leading to poor pod setting (Basandrai et al. 2005), whereas the late-sown crop flowered by mid-February and thus escaped low temperature stress resulting in optimum flowering and pod setting.

During the epidemic year 2004–2005, resistant ICCX 810800, moderately resistant (ICCV 90201) and moderately susceptible (C 235) cultivars produced much lower yields compared with that obtained in 2003–2004. Though the yield level was comparatively lower in the resistant cv. ICCX 810800, it still gave the highest yield ($1,204.8 \text{ kg ha}^{-1}$) in the early-sown crop, and then declined with the delay in sowing. No grain yield was obtained in highly susceptible cvs Pb 7 and ICCV 96029. This supports earlier results (Chongo et al. 2000a, b; Gan et al. 2006; Shtienberg et al. 2000) that showed under cool and wet conditions, application of foliar fungicides is required to realize optimum yield and quality even in resistant cultivars.

The low TDR during the year 2003–2004 may be attributed to the low weekly mean rainfall ($0.7\text{--}>15 \text{ mm}$ over 3 weeks) against $0.17\text{--}6.74 \text{ mm}$ over 9 weeks during the season (Fig. 10).

During 2003–2004 growing season, the average minimum and maximum temperature remained below 5°C and 21.5°C , respectively until 11 February. Subsequently, minimum and maximum temperature varied from 6 to $>10^{\circ}\text{C}$ and 23.8 to $>30^{\circ}\text{C}$ and 9.4 to 14.4 and 32.3 to 36.9°C from 12 February to 18 March and 19 March to 17 April, respectively. The maximum temperature varied from 15.8 to $>21^{\circ}\text{C}$ from 1 January to 25 February, 21 to $>28^{\circ}\text{C}$ from 26 February to 25 March and was below 33°C from 17 March to 17 April 2005. The minimum temperature varied from $<5^{\circ}\text{C}$ to $>13^{\circ}\text{C}$ during the growing season except during the period 8–21 January, when it was around 2°C (Fig. 11). It is evident that during the 2004–2005 growing season, maximum temperatures were favourable for disease development, and even the minimum temperature was higher and more favourable compared with the 2003–2004 growing season. During the 2004–2005 growing season mean maximum RH was $<90\%$ during 11 out of 15 weeks of active disease development, in contrast to only 5 weeks during 2003–2004 growing season (Fig. 12). Furthermore, the mean weekly minimum RH, 45.5--

68.4% during the period 5 February–25 March, 2005 was higher compared with $22.6\text{--}45.7\%$ during the same period in the 2003–2004 growing season (Fig. 12). Temperatures of $20 \pm 1^{\circ}\text{C}$, RH of $>90\%$ and leaf wetness of 17 h are optimum for the infection, development and spread of ascochyta blight (Pande et al. 2005, Trapero-Casas and Kaiser 1992). In addition, leaf wetness periods greater than 8-days results in the production of higher numbers of pycnidia and conidia on infected leaves (Jhorar et al. 1997). Such favourable conditions were prevalent in the controlled environment at ICRISAT and during the year 2004–2005 at Dhaulakuan, which led to severe disease development. Jhorar et al. (1997) observed that increased dry periods immediately after inoculation resulted in reduced disease severity and low disease development. Hence, low disease levels during the 2003–2004 growing season may be attributed to the continuous dry spell.

Blight severity in the controlled environment was higher and more consistent than under field conditions; this was because isolate AB 04 was more virulent than AB 06 (Basandrai et al. 2005) and environmental conditions were highly favourable and less variable than under field conditions.

The resistant and moderately resistant cultivars showed rate-reducing residual resistance against the virulent isolate AB 4, expressed as longer incubation periods, slower disease development and lower TDR. The highly resistant cv. ICCX 810800 and highly susceptible cvs Pb 7 and ICCV 96029 showed the same trend for ascochyta blight development at different growth stages under controlled environment and field conditions during the epidemic year. Hence, growth chamber and field screening under epidemic conditions at hot spots like Dhaulakuan are equally effective and may compliment each other.

All the cultivars used in the present study were developed in India, where *A. rabiei* is highly variable in virulence (Basandrai et al. 2005; Nene and Reddy 1987; Pande et al. 2005; Singh and Sharma 1998). Under such conditions, growing susceptible cultivars, namely Pb 7 and ICCV 96029, can result in total crop loss and even resistant cultivars such as ICCX 810800 can suffer heavy losses (Chongo and Gossen 2001; Chongo et al. 2000b; Pande et al. 2005). Efforts are being made to popularise chickpea cultivation in north western India. It will result in a substantial increase in the area grown to the crop. High levels of

resistance are not available against all pathotypes of *A. rabiei* in cultivated chickpea (Basandrai et al. 2005; Nene and Reddy 1987; Pande et al. 2005; Singh and Sharma 1998). Resistant cultivars such as ICCX 810800 still show reduced resistance at the flowering stage. Hence, for the successful cultivation of chickpea, integrated management of ascochyta blight using available resistant cultivars, disease-free seed and need-based foliar application of fungicides will be the practical option.

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